14.—Some Aspects of the Life Cycle of the Plant Pathogen Sclerotinia sclerotiorum in Western Australia

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Observations on the life cycle of *Sclerotinia sclerotiorum* were made in the Perth area in Western Australia during 1957 and 1958. The stages of the cycle of sexual reproduction observed agree with those reported from other parts of the world.

Ascospores are regarded as the only significant source of host plant infection.

Introduction

During investigations on diseases caused by the fungus *Sclerotinia sclerotiorum* (Lib.) D. By., 1886 in Western Australia during 1957 and 1958, particular emphasis was placed on establishing the life cycle of the causal organism as this would facilitate researches on methods of control. Although the cycle of sexual reproduction has not been observed previously in Western Australia, it is known in other parts of the world and the ascospores liberated from the apothecia known to be capable of host plant infection (Lauritzen 1932, Keay 1939, Purdy and Bardin 1953, Purdy 1958). However, Hungerford and Pitts (1953) and Purdy (1958) showed in the laboratory that sclerotia in soil may germinate to produce hyphae which can infect host plants at ground level. On the other hand, Keay (1939), Loveless (1951) and Henderson (1958) have been unable to produce infection consistently by this means.

This paper presents observations on the life cycle of *S. sclerotiorum* made in market gardens in the Perth Metropolitan area where the disease has become serious due to susceptible crops such as beans and cauliflowers having been grown intensively over a number of years.

The Life Cycle

The morphological stages of the life cycle which have been observed in the Metropolitan area are presented in Figure 1.

Depending on the form of post harvest cultivation, diseased plant residue containing sclerotia is either buried to varying depths in the soil or remains in the surface soil only. Sclerotia in the surface soil are a potential source of inoculum for the following crop. In an attempt to assess the sclerotial population in the surface soil, samples of soil were taken from several market gardens. In one garden, rotary hoe cultivation only had been carried out on one half of an area which had previously supported a diseased crop, while the other half had been deep ploughed. As many as twenty sclerotia were removed from soil samples (approximately 1 ft. square and 2 in. deep) which were taken from areas which had been rotary hoed, while a maximum of two only was obtained from samples from areas which had been deep ploughed (Table I). It is clear that with continued shallow cultivation there would be a build-up of sclerotia in the surface layers of the soil. The effectiveness of deep ploughing in reducing sclerotial numbers in the surface soil might become less significant with time due to an overall build-up of sclerotia unless the majority of sclerotia had only limited viability. Mr. S. C. Chambers of the Department of Agriculture in Perth is working on this aspect.

TABLE I

*The Concentration of Sclerotia in the Surface Two Inches of Soil on the Property of Mr. Schultz, Balcatta**

Post Harvest Cultivation	Sample	Number of Sclerotia Per Sample (approx. 1 ft. sq. and 2 in. deep)
Rotary hoeing only	1	12
	2	10
	3	20
Deep ploughing	1	2
	2	0
	3	2

* Sclerotia were removed from soil samples by the method used by Campbell (1946).

Viable sclerotia in the surface soil may germinate to form apothecia a few weeks after the onset of moist conditions. In the winter months the moisture is supplied by the natural rains and in the autumn, summer and spring by overhead sprinkler systems. Apothecia have been found in diseased crops throughout the whole year, though they are definitely more numerous during the winter months. From the time the crop is planted until post harvest cultivation there is continued and increasing production of apothecia in areas where the disease is prevalent. Field tests (Henderson 1958) have shown that sclerotia formed in a diseased crop in June can produce apothecia in a following crop in July, August, September and October. However, although the first apothecium was produced within five weeks, only approximately 10 per cent. had germinated by the end of October. It appears that under natural conditions, sclerotia germinate irregularly, some taking

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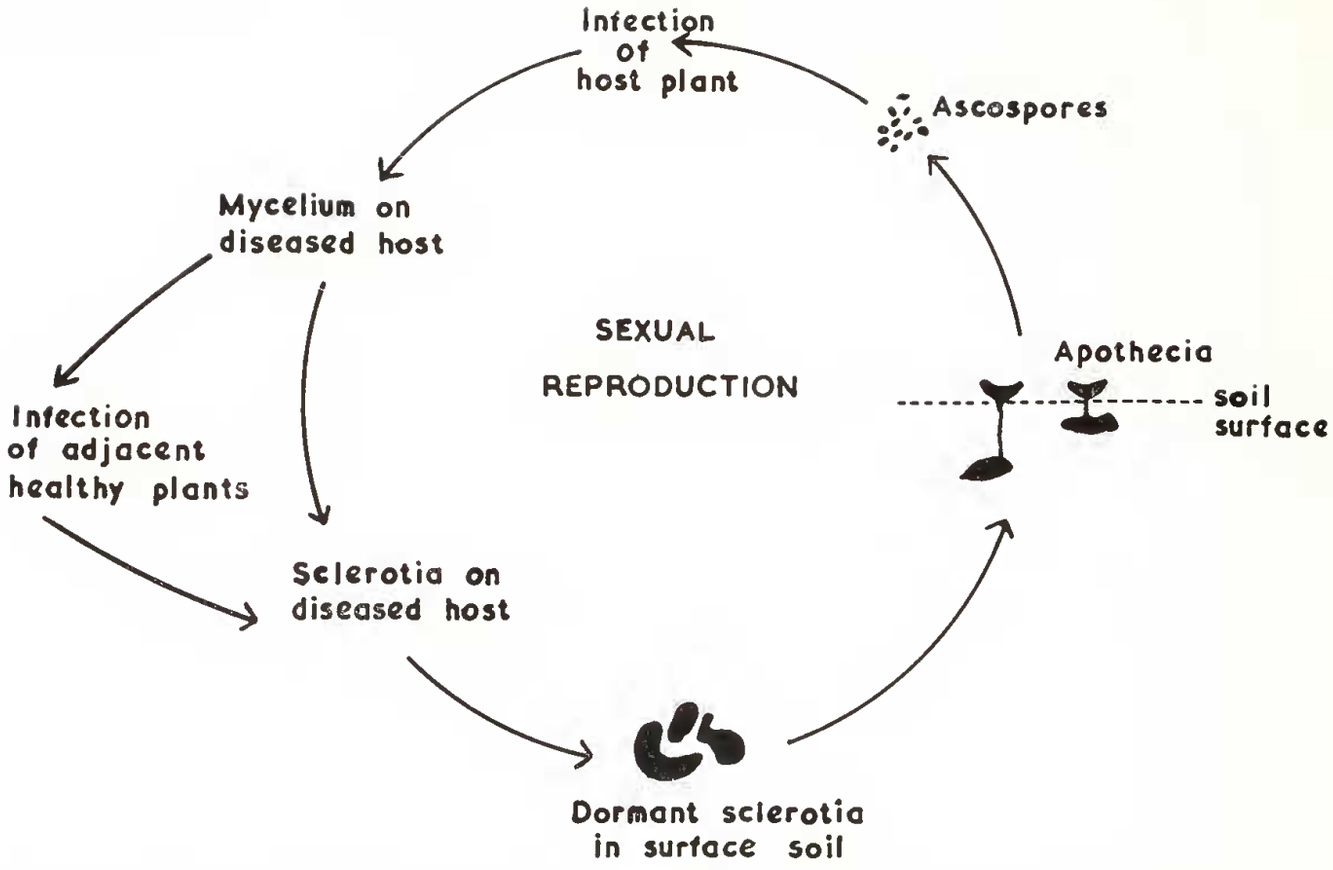

Fig. 1.—Diagrammatic representation of phases of the life cycle of *Sclerotinia sclerotiorum* observed in Western Australia.

much longer than others to produce apothecia. Tests were carried out by the author which showed that some sclerotia are capable of producing as many as three crops of apothecia over a period of four months (Henderson 1958).

In areas where the disease is prevalent a maximum average of nine apothecia-forming sclerotia has been found per square yard, each sclerotium producing one to several apothecia (Table II). As each apothecium may produce 30,000,000 ascospores (Ingold 1953) the number of ascospores released into the atmosphere may be very high. The ascospores are released from the apothecium over a number of days. Marked apothecia in the field have been observed to "puff" intermittently for a period of five to ten days.

Nowhere over a period of twelve months has a diseased crop been inspected without apothecia being found within the confines of the crop, or rarely, only in areas immediately adjacent to it. If basal infections were found, then apothecia were always observed in the immediate vicinity of the diseased plants. On the few occasions when only aerial infections were found, apothecia were not observed within the crop but were always easily found within neighbouring crops. It appears that after ascospores are liberated from the apothecia, they are carried by the wind onto adjacent host plants resulting in basal and aerial infections or rarely they may be carried further afield to neighbouring susceptible crops causing aerial infections only. Infection of host plant material with ascospores has been frequently reproduced in the laboratory.

TABLE II

Some Examples of the Density of Apothecia-forming Sclerotia in Market Gardens in the Metropolitan Area from April, 1957, to January, 1958.

Month	Property	Crop	Number of Apothecia-Forming Sclerotia Per Sq. Yd. (Av. of 10 Sq. Yd.)
April	Mr. Arbuckle, Balcatta	Cauliflower seedbed	2
,,	,, ,, ,,	Cabbage seedbed	1
,,	,, ,, ,,	Mature cauliflower	6
May	Mr. Sawle, S. Coogee	Mature bean	1·5
,,	Mr. Goodchild, Spearwood	,, ,,	1
June	Mr. Arbuckle, Balcatta	Mature cauliflower	6
,,	Mr. Goodchild, Spearwood	Young bean	3
,,	Mr. Donnetti, Balcatta	Mature bean	2
,,	Mr. Sawle, S. Coogee	Mature cauliflower	9
July	,, ,, ,,	,, ,,	2
Aug.	,, ,, ,,	,, ,,	1
Sept.	Mr. Di Piazzi, Osborne Park	Mature celery	A few only
Nov.	Mr. Arbuckle, Balcatta	Mature cauliflower	A few only
Jan. 1958	Mr. Donnetti, Balcatta	Mature lettuce	7 (Apothecia under plants only)

In Western Australia during 1957 and 1958 *Sclerotinia* disease was observed on cauliflowers, cabbages, brussels sprouts, beans, celery, lettuce, tomato and on the herb *Portulaca oleracea* which

is often found as a weed in the preceding crops. Records show that in Western Australia *S. sclerotiorum* may also infect potatoes, lucerne, W.A. blue lupin, N.Z. lupin, citrus, apricot, passion vine and Calendulas (unpublished data in W. Aust. Plant Disease Records, Agric. Dep. W. Aust.; Annu. Rep. Plt. Path., Agric. Dep. W. Aust.; File 367/47, Agric. Dep. W. Aust.)

Masses of white hyphae are formed on the host plant several days after infection. This mycelium subsequently produces the black sclerotial bodies which later fall to the ground in the diseased crop residue.

No asexual spores have been observed on mycelium on diseased plants which might have caused further spread of the disease. However, mycelium may grow from a diseased plant directly onto an adjacent healthy plant.

Conclusions and Diseussion

The stages of the cycle of sexual reproduction for *Sclerotinia sclerotiorum* observed in the Perth Metropolitan area, Western Australia agree with those found in other parts of the world.

It has been observed that there is a definite sclerotial population in the soil in areas where the disease is prevalent. These sclerotia are a potential source of inoculum whenever environmental conditions are suitable.

The presence of abundant apothecia in areas where the disease is prevalent implies that host plant infection is primarily due to ascospores. Furthermore, as no diseased crop with basal infections has been observed without apothecia being found on the soil in the immediate vicinity there has been no reason to suspect that any infections observed could have resulted from hyphae produced directly by sclerotia. The author (1958) has failed to produce this form of host plant infection in the laboratory except for one instance. This was in agreement with findings of Keay (1939) and Loveless (1951) but contradictory to the work of Hungerford and Pitts (1953) and Purdy (1958). The author considers that although hyphal infection by sclerotia may take place occasionally, it is not of practical signficance.

No asexual spores on host plant mycelium which might cause secondary infections have been observed.

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